

Variation In Resistance to White Pine Blister Rust Among 43 Whitebark Pine Families from Oregon and Washington – Early Results and Implications for Conservation

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Abstract

All nine North American species of white pines are susceptible to the introduced, invasive pathogen *Cronartium ribicola*, the cause of white pine blister rust. Whitebark pine is considered one of the most susceptible species. Genetic resistance is considered a cornerstone for survival to this pathogen. Fortunately, all of the native species of white pines have some level of resistance. Evaluation of resistance in Oregon and Washington families of whitebark pine has only recently begun; currently over 150 seedlots collected from individual parent trees are in resistance testing. This report summarizes differences in responses among 43 seedling families and one bulked seedlot through two years after artificial inoculation with blister rust. Initial infection after inoculation of three-year-old seedlings was very high in the first set of trials; 100% of the seedlings developed needle lesions in the two trials reported in this paper. There were large differences among families in several traits, including percentage of trees with stem symptoms and survival two years after inoculation. The level of resistance present in some families and the frequency of resistance among the 43 families reported here is encouraging. A possible geographic trend in resistance is also noted. It is recommended that at least a subset of families be field planted to validate resistance ratings from this short-term screening. The collection and use of seed from putative resistance parent trees identified through this testing would be a good starting point for restoration efforts.

Introduction

Whitebark pine (*Pinus albicaulis* Engelm.) is one of the nine native species of white pines (also known as five-needle pines) in the U.S. and Canada. All nine species are susceptible to white pine blister rust, a disease caused by the non-native, invasive pathogen *Cronartium ribicola* (J.C. Fisch.). Whitebark pine is considered to be among the most susceptible of the nine North American species of white pines (Hoff and others 1980).

Concerns about the status of whitebark pine in Oregon and Washington began to escalate in the 1990s (Delprato 1999; Snieszko and others 1994). Since then a series of additional field surveys have been undertaken (e.g. Erickson and others, this proceedings; Goheen and others 2002), and an overall summary of these reports has recently been prepared (Ward and others 2006). Blister rust infection and mortality are having heavy impacts on whitebark pine and its associated ecosystems throughout the range of the species, and concerted action is needed

to revitalize these areas (Schwandt 2006). The concerns about the whitebark ecosystems in Oregon and Washington led to the start of a four-year (2005 to 2008) 'Albicaulis Project' to further assess the status of this species, to organize additional cone collections, and to develop a conservation plan (see <http://www.fs.fed.us/r6/genetics/programs/whitebark-pine/albicaulis/> for details).

Fortunately, there is at least a low frequency of naturally-occurring genetic resistance in whitebark pine (Hoff and others 2001). Such genetic resistance has been used as a cornerstone for potential restoration and reforestation efforts for other species (Snieszko 2006) and is viewed as an essential element in such efforts with whitebark pine (Samman and others 2003; Schwandt 2006). Cone collections from National Forests, National Parks, and the Confederated Tribes of Warm Springs were started in the mid-1990s to use in rust resistance testing and in studies of genetic variation (For results from early common garden genetic studies see Bower 2006; Hamlin and others, this proceedings.).

The Pacific Northwest Region of the USDA Forest Service (Region 6) has begun testing seedling families of whitebark pine for resistance to white pine blister rust. Over 150 families are now being tested at Dorena Genetic Resource Center (DGRC), and several hundred additional seedlots are scheduled to be sown in early 2007 for testing (many of these latter collections were funded through the 'Albicaulis' project). These seedlots represent much of the range of whitebark pine (WBP) in Oregon and Washington (Region 6) (Table 1). Resistance screening of these seedlots will provide baseline data on levels, frequencies and patterns of resistance. Options for conservation and restoration strategies for affected areas will depend upon the level and frequency of resistance and any geographical trends in resistance.

Table 1. Number of Oregon and Washington whitebark pine seedlots sown for operational blister rust screening at Dorena Genetic Resource Center by year.

Organization	# Families Tested by Sow Year ^a					totals
	2001	2002	2004	2005	2007	
Deschutes NF		10		3	47	60
Fremont NF	4	14		7	15	40
Gifford Pinchot NF					14	14
Malheur NF			19		8	27
Mt. Baker-Snoqualmie NF					4	4
Mt. Hood NF		1	19		3	23
Okanogan NF					10	10
Olympic NF					9	9
Umatilla NF		1	27		2	30
Umpqua NF					10	10
Wallowa-Whitman NF					13	13
Wenatchee NF					38	38
Winema NF		4			10	14
Colville NF		13		5	11	29
Crater Lake NP			10		25	35
Mt. Rainier NP			19	9	4	32
Warm Springs CT			7		12	19
Rogue River NF (Mt. Ashland)					1	1
totals	4	43	101	24	236	408

^a Operational trials at DGRC are identified by the year sown; some seedlots have been included in more than one trial. Inoculation of seedlings occurs in September two or three years after sowing.

Inoculation and assessment protocols for whitebark pine have been adapted from those used for over 40 years in testing for resistance in western white pine (*P. monticola* Dougl. ex D. Don.) and sugar pine (*P. lambertiana* Dougl.) in Region 6 (see Kegley and Sniezko 2004 for some background) and refined in a small prototype test with whitebark pine (see Kegley and others 2004). The first operational inoculation of seedling families from 43 candidate trees selected in Region 6 was conducted in September 2004. Some of the early results are reported here; the seedlings will continue to be assessed over the next three years. Additional operational tests will be reported in future years (see Table 1 for other tests underway).

Objectives of this first operational screening were to (1) confirm the transferability of the rust testing protocols utilized for sugar pine and western white pine programs to whitebark pine; (2) produce the first estimates of the frequency and types of resistance in whitebark pine from Region 6 parent trees; (3) examine the influence of two inoculum sources of the pathogen on resistance; (4) compare the relative level and types of resistance of whitebark pine and western white pine; and (5) discuss some of the implications of the findings to whitebark pine conservation. Only the early assessments of these trials were available for this conference – updates will be provided upon final assessment.

Materials and Methods

Plant Material

The first significant inoculation of individual Region 6 whitebark pine (WBP) families occurred in 2004. This trial included a total of 43 WBP families (seedlings from wind-pollinated parent trees located in natural stands) from six different National Forests in Region 6 as well as a bulked WBP seedlot from the Shoshone National Forest (Forest Service Region 2). The bulked Shoshone lot (CDA # 7425) included seed of >50 parent trees southwest of Dubois, WY (~43°30'N ~109°50'W, elevation ~2987 m) (E. Jungck, pers. comm.). There were 2 groups of Shoshone material; one group of seedlings had been grown at the USDA Forest Service nursery in Coeur d'Alene, Idaho, and another group was sown and grown at DGRC with the Region 6 families.

The DGRC-grown seedlings were germinated in March 2002 following the protocols described by Riley and others (this proceedings) and cultured in supercell containers (16.4 cm³) for two growing seasons (see Riley and others, this proceedings). The WBP seedlings were grown in non-replicated family plots for the first two growing seasons and were a composite from several culturing treatments. The seedlings were transplanted into standard wooden frames (0.91 m wide x 1.21 m long x 0.30 m high) at DGRC approximately 14 months before inoculation. Seedling families were randomized across six replications in 10-tree row plots; some families had less than 60 seedlings and were distributed across the replications accordingly. Eight family row plots were included in each frame. The Idaho-grown Shoshone seedlot was represented in four plots in each replication.

In addition to the whitebark pine, eighty seedlots of western white pine were stratified, then sown directly into standard wooden frames at DGRC in March 2003, and cultured for two growing seasons. Both open-pollinated and control-pollinated seedlots were included and represented an array of susceptible and resistant types. As with the whitebark pine, the western white pine (WWP) families were randomized into six replications of up to 10 trees per replication in family row plots (see Kegley and Snieszko 2004 for a description of the basic study design for WWP operational blister rust screening trials). Two WWP families in this trial, one susceptible and one resistant control, were used to compare relative resistance of WBP with WWP.

Inoculation

Two separate trials, each utilizing a different inoculum source, were conducted. Leaves of *Ribes* spp., the alternate host for blister rust, infected with *C. ribicola* at the telial stage were collected for the inoculations. For **Trial 1**, three of the six replications of both WBP and WWP were inoculated concurrently during the week of August 23, 2004 with a source of rust with known virulence to a specific major gene (Cr2) in WWP. Cr2 conditions a hypersensitive reaction (HR) in the needles of WWP (Kinloch and others 2003), and the virulent source of rust (vcr2 source) would be expected to render HR in WWP ineffective (see Kinloch and others 2004 for discussion). In the absence of vcr2, the WWP resistant control is expected to show only 25% of seedlings with stem symptoms. Approximately one week later, three different replications were inoculated (**Trial 2**) with a composite collection of inoculum from several areas outside the geographic areas where vcr2 has been previously documented (Kinloch and others 2004) (AVCr2 source). Trial 1 used *R. bracteosum* Dougl.

ex Hook. collected on the Umpqua National Forest Cottage Grove Ranger District and *R. nigrum* L. and *R. hudsonianum* Richards. var. *petiolare* from the DGRC Ribes Garden. Trial 2 used *R. hudsonianum* var. *petiolare* leaves from eastern Oregon (Wallowa-Whitman, Malheur, and Ochoco NF), central Oregon (Silver Lake), and southern Washington (Gifford Pinchot NF and Ahtunum Creek, which is near existing WBP stands) and *R. bracteosum* from Trout Lake, WA.

Inoculation followed standard DGRC procedure (Danchok and others 2004). The target inoculum density for both species and both inoculations was 3000 basidiospores/cm², which is the standard inoculum density for operational screening of WWP at DGRC. Actual inoculum densities were 2946 and 3482 basidiospores/cm² for Trial 1 and Trial 2, respectively (std err = 73 and 90, for Trial 1 and 2).

Disease Assessment and Analysis

Seedlings were assessed for survival and the presence and number of blister rust symptoms. Number of needle lesions ('spots') was assessed in May 2005 (8.5 months after inoculation). Presence of spots as well as number and type of stem symptoms were assessed in December 2005 (15.5 months after inoculation). Mortality was assessed in early August 2006 (24 months after inoculation). Third-year height (height at time of inoculation) was measured in May 2005. This paper reports percent spotting (% SPOT), number of spots at 8.5 months after inoculation (SPOT1#), percentage seedlings with stem symptoms (% SS2), number of stem symptoms per infected tree (SS2#), percentage infected seedlings surviving (% RSURV2), and percentage seedlings surviving with stem symptoms (% SSAL2).

Only preliminary analyses of the untransformed data have begun; analyses of variance were performed using plot means using SAS Proc GLM, and Pearson product-moment correlations were calculated using family means (SAS Inc. 2006). Trees dead from non-rust causes (notably due to *Phytophthora* spp.) were excluded from the dataset. Family identity information was compromised in one box of eight families in Rep 3, and those observations were also excluded from the analysis.

Results

Whitebark Pine – General Trends

Inoculation was very effective. 100% of the WBP seedlings had needle infections at the May 2005 assessment (8.5 months after inoculation), and the majority (93.9 and 90.0% for the Trial 1 and Trial 2, respectively) still had needle lesions present seven months later, 15 months after inoculation (% SPOT2, Table 2). There were significant differences among families ($F = 3.76$, $p < 0.0001$) and replications ($F = 21.5$, $p < 0.0001$) for number of spots. Number of spots averaged 14.9 and 18.9 for Trials 1 and 2, respectively (SPOT1#, Table 2), while family means ranged from 9.4 to 24.9 over the two trials. In general families tended to perform similarly in both trials (Figure 1, $r = 0.57$, $p < 0.0001$).

Table 2. Population (forest) means for whitebark pine (WBP) and family means for western white pine (WWP) controls after inoculation with two inoculum sources (Trial 1 and Trial 2).

Group	# families	spot1# ^a		% SPOT2 ^b		% SS2 ^b		% RSURV2		% SSAL2	
		Tria 1 1	Trial 2	Tria 1 1	Tria 1 2	Tria 1 1	Tria 1 2	Tria 1 1	Tria 1 2	Tria 1 1	Trial 2
All families	48	14.9	18.9	93.9	90.0	87.7	89.8	25.6	24.4	18.3	18.1
Deschutes	10	15.5	18.2	91.1	90.2	92.4	95.2	19.7	12.1	17.5	8.1
Fremont	14	17.1	21.2	91.8	90.1	100.0	97.3	8.8	5.1	8.8	2.7
Mt Hood	1	18.1	18.4	96.7	93.0	45.6	82.0	82.6	81.5	61.9	77.5
Umatilla	1	10.7	15.3	81.7	66.7	100.0	91.7	4.2	0.0	4.2	0.0
Winema	4	13.9	21.1	97.5	96.4	100.0	94.1	6.8	15.8	6.8	11.4
Colville	13	13.1	19.4	97.7	96.6	69.6	76.3	55.3	58.5	35.8	46.0
DGRC-grown Shoshone	1	19.6	16.1	100.0	83.3	85.0	100.0	20.6	10.0	5.6	10.0
CDA-grown Shoshone	4	12.0	11.0	92.8	67.6	87.8	87.8	14.2	15.4	4.5	4.2
WWP susceptible	1	--	--	93.3	93.3	100.0	93.3	100.0	100.0	100.0	100.0
WWP MGR	1	--	--	74.8	53.3	92.6	26.7	96.7	100.0	100.0	100.0

^a 100% of the WBP seedlings and the WWP controls had spots ~8.5 months after inoculation. Actual number of spots was not counted for the WWP; relative number of spots was indicated by assignment into classes (see Kegley and Snieszko 2004 for details of the procedure)

^b Adjusted to exclude Phytophthora mortality

The percentage of seedlings with stem symptoms (% SS2) was similar for the two trials, 87.7% and 89.8% for Trials 1 and 2, respectively. There were significant family and replication differences for % SS2. Family means varied from 39.9 to 100% in Trial 1 and from 23.3 to 100% in Trial 2 (Figure 2). Fifteen of the 43 seedling families showed 100% SS2 in both Trial 1 and 2. Families tended to perform similarly with both inoculum sources ($r = 0.75$, $p < 0.0001$), but a few families showed differences of 30% or more (Figure 2). In general, the Colville families and the single Mt. Hood family had fewer seedlings with stem symptoms relative to the other populations (Table 2, Figure 2). One of the Deschutes families also showed relatively low % SS2. Infection (presence of needle spots or stem symptoms) was 100% in both trials.

Figure 1. Family mean number of spots per seedling (SPOT1#) in Trial 1 (vcr2 inoculum source) vs. Trial 2 (AVCr2 inoculum source) for seedlots from 7 National Forests

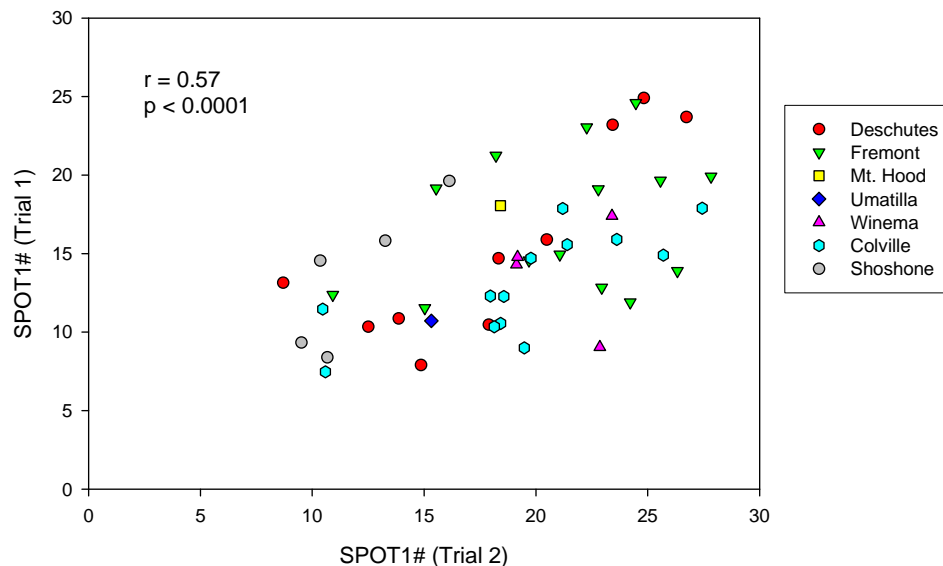
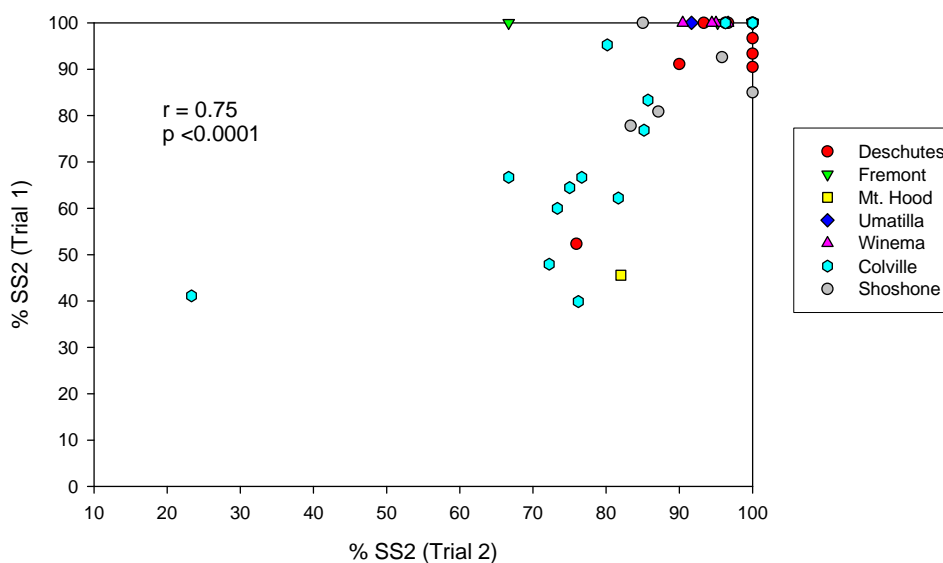
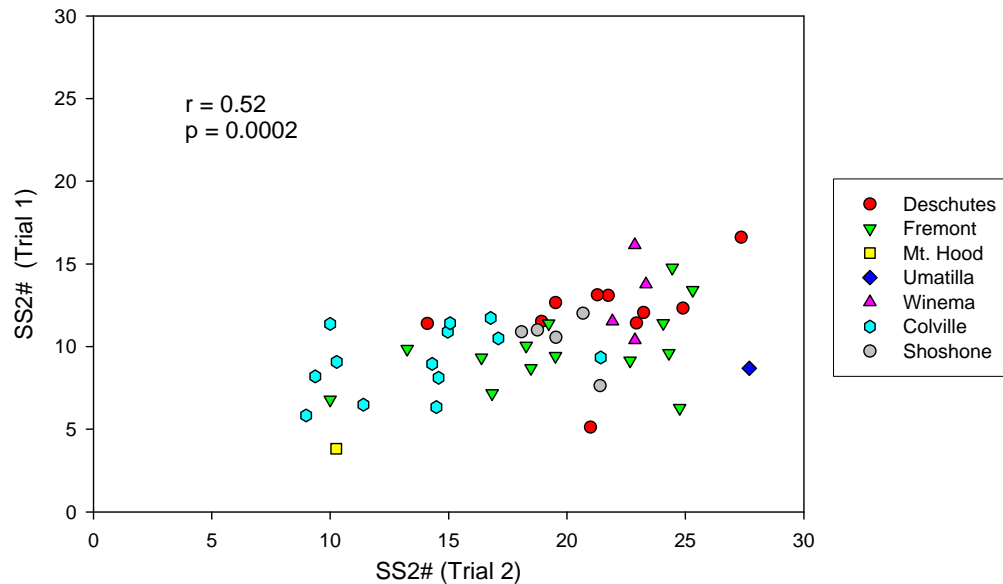


Figure 2. Family mean percentage seedlings with stem symptoms (% SS2) in Trial 1 vs. Trial 2 for seedlots from 7 National Forests



Although overall % SS2 was similar across the two trials, there were significant family, inoculum source, and replication differences in number of stem symptoms per infected seedling (SS2#); mean SS2# was 10.2 for Trial 1 and 18.7 for Trial 2; family mean SS2# ranged from 3.8 to 16.6 for Trial 1 and from 9.0 to 27.7 for Trial 2 (Figure 3). The Mt. Hood family and many of the Colville families had among the lowest SS2#, while the four Winema families had among the highest SS2# in both trials (Figure 3).

Figure 3. Family mean number of stem symptoms (SS2#) in Trial 1 vs. Trial 2 for seedlots from 7 National Forests



Survival of seedlings with stem symptoms two years after inoculation (% SSAL2) averaged around 18% for both trials (Table 2); family means ranged from 0 to nearly 75% for the two trials. Fourteen of the 43 families had at least 15% SSAL2 in both trials, and four of these families had more than 50% SSAL2 in both trials. It was noted that, at least for the first two years after inoculation, the families that had lower % SS2 also tended to have higher % SSAL2 (Figure 4).

Survival of infected seedlings nearly two years after inoculation (% RSURV2) was similar between the two trials, averaging 24.5% for Trial 1 and 22.4% for Trial 2 (Table 2, Figure 5). Dramatic differences in survival were evident among families (Figures 5 and 6a). Family mean survival ranged from 0 to 81.5% for Trial 1 and from 0 to 90% for Trial 2 (Figure 5). Four of the 43 Oregon and Washington families had no survivors in either trial. Two years after inoculation, 13 of the 43 seedling families (30% of families) showed moderate to high levels of survival (>35% seedlings alive), including 11 of the 13 Colville families (Figure 5). The remaining 30 families and the Shoshone bulk lot generally showed much lower levels of survival. Families tended to show similar levels of survival in both trials (Figure 5, $r=0.87$, $p<0.0001$).

Whitebark Pine versus Western White Pine

The WWP susceptible control exhibited 100% and 93.3% SS in Trial 1 and Trial 2, respectively (Table 2). The MGR WWP control family had 92.6% SS in Trial 1 and 26.7% SS in Trial 2 (Table 2). This WWP family was expected and did exhibit a differential response to the two inoculum sources (higher % SS when inoculated with the vcr2 source); the susceptible WWP family and the WBP families did not exhibit this differential response. The susceptible WWP averaged 7.7 and 11.1 SS per infected tree for Trial 1 and Trial 2 respectively. This is slightly lower than the trial averages for all WBP families, despite the WWP being much larger in size. Third-year seedling height of the WBP averaged 9.2 cm in Trial 1 and 8.8 cm in Trial 2; family means ranged from 5.0 to 13.6 cm across the two trials;

whereas the WWP controls averaged between 27.1 and 29.4 cm. Through August 2006, no mortality had occurred in these two WWP seedlots compared with high mortality noted in the whitebark pine (Figure 6b).

Figure 4. Family mean % survival with stem symptoms (% SSAL) vs. percentage seedlings with stem symptoms (% SS) for Trial 1 and Trial 2 for seedlots from 7 National Forests

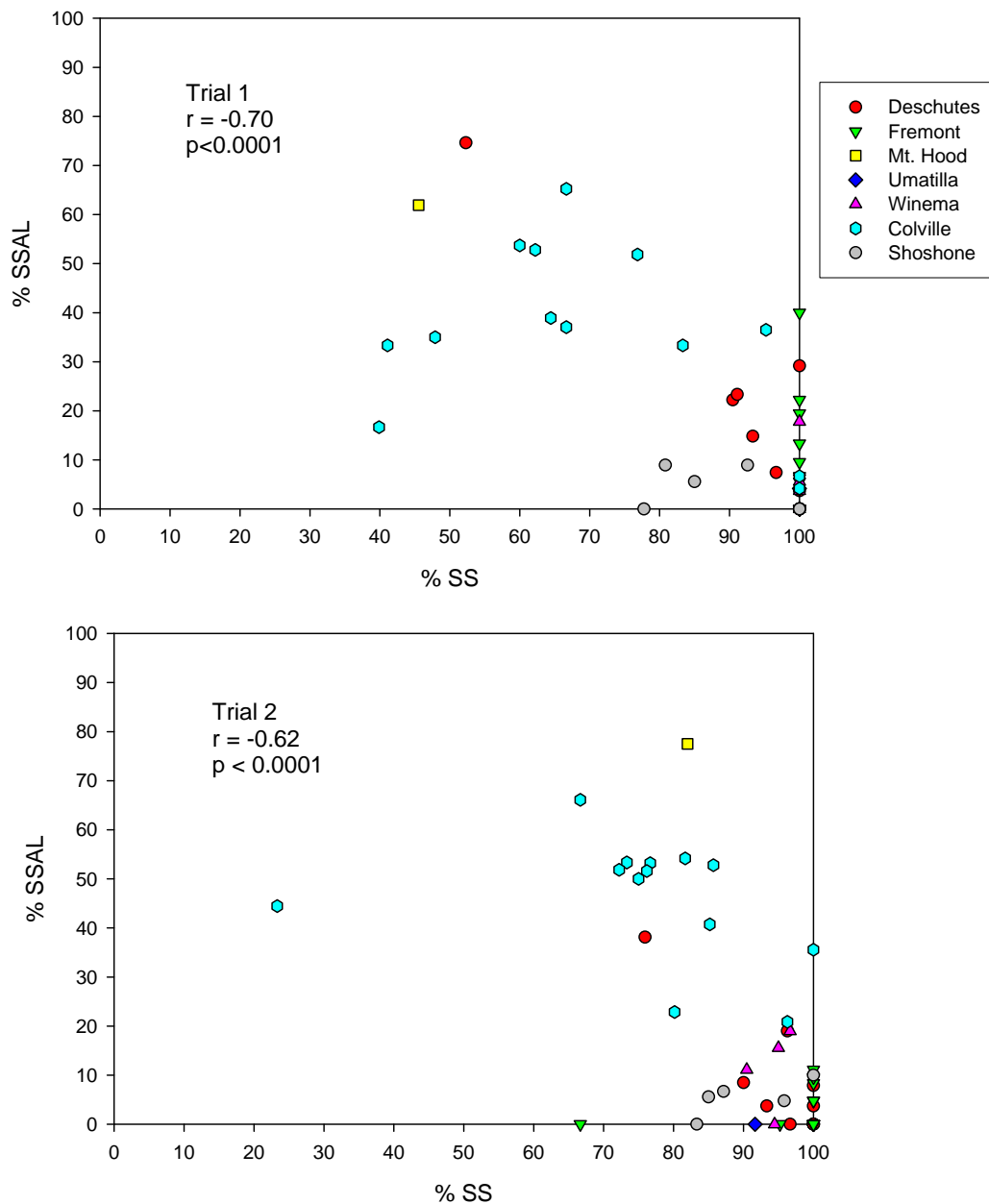


Figure 5. Family mean % survival of infected seedlings (%RSURV2) in Trial 1 vs. Trial 2 for seedlots from 7 National Forests

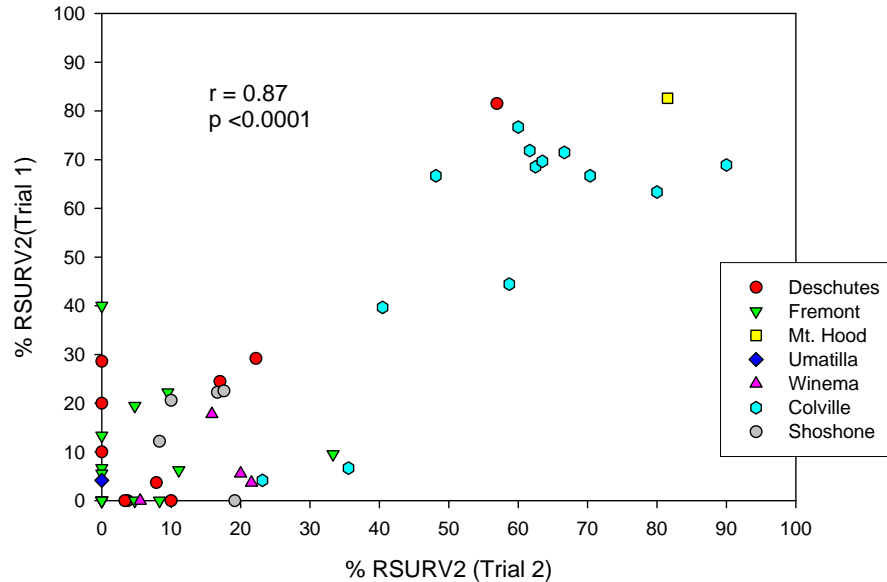


Figure 6. (a—Left)) Survival and mortality (red trees) of WBP in May 2006. Note the high survival of one family from the Colville NF. (b—Right) Comparison of WWP (tall green trees) and WBP



Discussion and Summary

It will still be several years before we have final results from these two trials, but at this stage it appears that there is little practical difference between the two inoculum sources used. Similar results were found in a smaller, earlier trial using two different geographic sources of inoculum; the % SS and % survival were similar between the two inoculum sources (Kegley and Snieszko 2004; Kegley and others, this proceedings). If this result holds for the trials presented here, it would imply that easily accessible sources of inoculum could be used to evaluate whitebark pine for resistance. However, the ultimate validation will come from field tests.

The moderate to high frequency of families with low % SS in the 43 field selections tested is higher than that found our rust tests of field selections of WWP or sugar pine (see Kegley and Sniezko 2004). However, the WBP seedlings averaged more SS per tree than the susceptible WWP control, and mortality in WBP proceeded more rapidly than in WWP. The rapid mortality of seedlings with stem symptoms may be a function of several factors, including seedling size or intrinsic susceptibility of the species. From our experience at DGRC, WBP, WWP, sugar pine, and eastern white pine (*P. strobus* L.) are all very susceptible, with sugar pine and eastern white pine showing faster mortality than WWP. However, even through this stage of assessment, the level and frequency of resistance (notably low % SS) found in this first test of Region 6 families was somewhat unexpected.

The Shoshone seedlot was a bulked collection and had been included in an earlier prototype trial to examine the effect of inoculum density and geographic source of inoculum on WBP (Kegley and others 2004; Kegley and Sniezko, this proceedings). As in that earlier trial, the Shoshone seedlot also showed relatively low levels of resistance and low survival (10 and 11.8% for DGRC-grown and Coeur d'Alene-grown seedlings, respectively). In another test, this seedlot ranked 59th for rust resistance among 108 seed sources from the Inland Northwest, using an index of resistance traits (Mahalovich and others 2006).

The level of infection, particularly the number of stem symptoms per seedling, is high in these two trials and should represent a good test of resistance. However, this high level of infection may overwhelm some types of partial resistance in small seedlings, leading to high mortality in some families. In addition some resistant traits (e.g. low # stem symptoms) may not be as effective in very young seedlings as they would be in the field on larger trees. Thus, further examination of traits such as the number of needle spots, number of stem symptoms and time of mortality is needed. Field validation of the effectiveness of these traits is also essential.

The number of resistance mechanisms present in WBP is unknown and may vary by geographic origin. Hoff and others (2001) summarized resistance work in several populations of WBP and found three of the putative resistance mechanisms that have been reported in WWP of Idaho origin: 'Needle shed' and 'Short-shoot', resistances which prevent stem infection, and 'Bark Reaction', a resistance that produces an incompatible stem infection. In a larger study of sources from the Inland Northwest, resistance responses similar to those reported by Hoff were found (Mahalovich and others 2006). In the two DGRC trials reported here, most trees still had spots at the second assessment (15.5 months after inoculation), by which time they were cankered. In the DGRC trials, the few families with lower % SPOT2 had very high levels of stem symptoms and mortality (unpublished data). The underlying mechanism resulting in a no stem symptom phenotype in Oregon and Washington WBP and its inheritance is currently unknown. Through nearly two years after inoculation, the bark reaction response was very infrequent and incomplete (partial) in our trials.

In this study of progeny of Oregon and Washington trees, apparent differences in SPOT1#, SS2#, % SS2, % SSAL2, and % RSURV2 were observed, and further examination of these traits is underway. In general, the families showing the lowest % SS2 were also the families

showing the highest % SSAL2. The parents of these families may have several resistant mechanisms, or seedlings within the families may manifest the resistance slightly differently. These are also the families with the highest survival through August 2006 assessment. These results are still preliminary, especially for the survival traits. Many infected seedlings are heavily cankered but currently alive, SSAL (Figure 7a and 7b), and are expected to die in the next two years.

From the modest number of seedlots sampled here, one of the four geographic areas with multiple families stands out for resistance, the Colville NF. Most of the Colville families had 40 to 80 percent SS and were in the top quartile for survival. In addition, the one seedlot from Mt. Hood NF also was showing high survival at this stage. This early data suggests that in Oregon and Washington, resistance may be in higher frequency in the northern part of the geographic range. This possibility is under further investigation in other trials (Table 1). In a study of Interior Northwest seed sources, principally from Idaho and Montana, an increase in some types of resistance was noted from southeast to northwest (Mahalovich and others 2006). Four families from Colville NF were included in that trial, and two were among the top 10 ranked for resistance and represented the northwestern part of the range sampled in that study.

The higher incidence of rust resistance noted in collections from the northern areas in this study may be due to several factors, including higher incidence of rust in the northern part of the range. At this point in time, information about infection levels from all of the parent tree stands is incomplete. However, recent surveys have noted that northern stands generally have greater levels of rust infection (Shoal and Aubry 2006). The higher incidence of rust in the north would have led to higher rust mortality, thus leaving a higher percentage of trees with rust resistance (see Hoff 1994 for discussion). Hoff (1994) observed higher levels of resistance in WBP families from stands with high mortality due to blister rust and lower levels of resistance in WBP from stands with low to moderate mortality (44% canker-free vs. 4% and 18%, respectively). The Region 6 WBP parent trees represented by seedling families in this test were selected on the basis of cone availability and not necessarily for blister rust resistance. Further work is needed to examine the rust incidence and mortality in areas where seed collections were made.

Recent research suggests that whitebark pine is most closely related to the Eurasian stone pines and is likely the result of migration through the Bering Strait opening more than 1.8 million years ago (Krutovskii and others 1994; A. Liston, pers. comm.). The Eurasian stone pines have shown very high blister rust resistance in screening trials (Hoff and others 1980). It is possible that the retention of at least some ancestral rust resistance genes may help explain the relatively high frequency of resistance in northern populations of WBP. Additional rust screening trials will help clarify the pattern of resistance in WBP.

Short-term resistance screening can be a very valuable tool to efficiently evaluate many parents for resistance. Additional work currently underway with whitebark pine includes examining many additional families for resistance, testing some of the highest surviving families for HR (not yet documented in WBP), examining the effect of inoculum density on resistance, and determining whether even younger seedlings can be used to rate families for

resistance (which could reduce costs and shorten the time period for assessing resistance). However, planting of a subset of seedling families on whitebark pine sites will be essential to confirm that this resistance is also effective under field conditions and to monitor the durability of resistance for this species.

The parent trees of the families rated for resistance in this test can be used as ‘permanent plots’ to follow the continued progression of rust as well as to monitor the durability of field resistance. Consideration should be given to protecting some of these parent trees from mountain pine beetle (*Dendroctonus ponderosae* Hopkins) so that they can serve as both cone producers and as monitors of field resistance. Parent trees of seedling families showing good survival, particularly those showing the lowest levels of SS%, are candidates for seed collection or inclusion in seed orchards (seedlings within these families are also candidates for inclusion in orchards). Families performing well for other traits (for example, families with few stem symptoms) should also be considered. However, additional families will have to be evaluated for resistance to increase the genetic base of any orchards established.

Conservation and restoration possibilities for whitebark pine will depend on a variety of factors, but the frequency and durability of resistance and rust hazard of the site will be two of the most important. Establishing resistant seedlings will be a key step in moderate to high disease areas. Encouraging abundant natural regeneration can also be an important step in maintaining whitebark pine in ecosystems (Schoettle and Snieszko, in press; Schoettle and others, this proceedings). Further development of seed transfer guidelines for whitebark pine within Oregon and Washington would be valuable in helping transfer resistant seed from one area to other areas where there is little or no resistance.

Figure 7. Seedlings surviving with multiple stem symptoms (January 2007)



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